AMENDMENTS TO THE SPECIFICATION

On page 32, please replace the first paragraph with the following rewritten paragraph.

-- Figure 1 depicts the DNA and deduced amino acid sequences (SEQ ID NOs: ~~50-51~~ 66-67) of an NMSup35-GR chimeric gene described in Example 1. --

On page 41, please replace line 23 to page 42, line 10 with the following rewritten paragraph.

-- A chimeric polynucleotide Fig. 1 and (SEQ ID NO: ~~50~~ 66) was constructed comprising a nucleotide sequence encoding the N and M domains of Sup35 (Fig. 1 and SEQ ID NO: ~~50~~ 66, bases 1 to 759) fused in-frame to a nucleotide sequence (derived from a cDNA) encoding the rat glucocorticoid receptor (GR) (Genbank Accession No. M14053, Fig. 1 and SEQ ID NO: ~~50~~ 66, bases 766-3150), a hormone-responsive transcription factor, followed by a stop codon. This construct was inserted into the pRS316CG (ATCC Accession No. 77145, Genbank No. U03442) and pG1 (Guthrie & Sink, "Guide to Yeast Genetics and Molecular Biology" in *Methods of Enzymology*, Vol. 194, pp. 389-398 (1981)) plasmids under the control of either the CUP1 promoter (plasmid pCUP1-NMGR, inducible by adding copper to the growth medium) or the constitutive GPD promoter (plasmid pGDP-NMGR). The nucleotide sequences of CUP1 and GDP (Genbank Accession No. M13807) promoters are set forth in SEQ ID NOs: 11 and 48, respectively. The GR coding sequence without NM, in the same promoter and vector constructs (plasmids pCUP1-GR and pGDP-GR), served as a control. GR activity in transformed yeast was monitored with two reporter constructs containing a glucocorticoid response promoter element (GRE) [Schena & Yamamoto, *Science, 241*:965-967 (1988)] fused to either a β-galactosidase (Swiss-Prot. Accession No. P00722) or to a firefly luciferase (Genbank Accession No. M15077) coding sequence. When GR is activated by hormone, *e.g.*, deoxycorticosterone (DOC), it normally binds to the GRE and promotes transcription of the reporter enzyme in either mammals or yeast. See M. Schena and K. Yamamoto, *Science 241*:965-967 (1988).

On page 61, please replace the paragraph that begins on line 8 with the following amended paragraph:

In another alternative embodiment, known prion sequences or other SCHAG amino acid sequences are modified, *e.g.*, by addition, deletion, or substitution of individual amino acids; or by repeating or deleting motifs known or suspected of influencing fibril-forming propensity. To form novel prion sequences, modifications to increase the number of polar residues (glutamine, asparagine, ~~serine~~ serine, tyrosine) are specifically contemplated, with modifications that increase glutamine and asparagine content being highly preferred. [See Depace *et al., Cell, 93*:1241-1252 (1998), incorporated herein by reference.] In a preferred embodiment, the alterations are effected by site directed mutagenesis or *de novo* synthesis of encoding polynucleotides, followed by expression of the encoding polynucleotides.

AMENDMENTS TO THE SEQUENCE LISTING

Please replace the sequence listing of the application as filed (pages 1-68) with the substitute sequence listing submitted herewith (pages 1-77).